ORIGINAL ARTICLES

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MYOCARDIAL INSUFFICIENCY.*

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In approaching this subject the first thing to consider is, what are the essential symptoms of myocardial insufficiency. While it is necessary in every case to recognize the evidence afforded by careful physical examination supplemented by the use of mechanical aids, it must be borne in mind that the facts when revealed do not give the information which is essential. The essential question for the physician to decide is whether the symptoms indicate the presence of an insufficient myocardium and the likelihood of heart failure. This knowledge can only be acquired in many cases by understanding what happens when the individual is engaged in some occupation that calls for some extra effort on the part of the heart. Hence all means of observation employed when the body is at rest can only yield very incomplete evidence. The kind of evidence which is essential is to be sought for in the symptoms evoked when effort is made, frequently only evoked then; as well as in the symptoms of functional insufficiency when the body is at rest. These symptoms are often so elusive and so insignificant that they readily escape the attention, while it is often difficult to appraise their value when detected. As our knowledge of what is meant by heart failure increases, the real significance of such symptoms will be revealed. Heart failure may be defined as the condition in which the heart muscle is unable to maintain an efficient circulation during the efforts necessary to the daily life of the individual.

Attacks of angina pectoris constitute a very definite clinical picture with which you are all quite familiar. Typically there is a sudden severe pain situated behind the lower sternum radiating perhaps to the neck or left shoulder or to the inner side of the left arm, associated with a strong sense of constriction of the chest, so that the breath is caught with difficulty and with a sense of great weakness and anxiety so that the patient feels that he will surely die. At the time of such an attack the patient is prostrated. The pulse may or may not be increased in rate but is often irregular. These attacks are very typical and are not difficult to recognize, either when they are seen or when the patient tells you about them, and I believe that to such attacks as these the term angina pectoris should be confined. Then we have a train of symptoms associated with coronary disease which differ somewhat from those of true angina. The pain, which is usually less severe, is a mid-line pain. Its characteristic seat is behind the lower sternum opposite the third and fourth intercostal spaces, at the xiphoid or in the epigastrium opposite the fifth intercostal space. Rarely it is felt as high as the second intercostal space. When severe it radiates and passes laterally to the left or right at these levels and may seem to pass through to the back. In the lesser manifestations or coronary disease seen early, there may be no pain at all but merely a sense of discomfort, felt especially under the lower sternum or in the epigastrium and not as the patient will say, "in the stomach." It is never a constant sensation, but comes in attacks, and these attacks either arise spontaneously when the patient is at rest or come on as the result of exercise, so that the patient must stop and rest until the discomfort passes off. It has a very peculiar character best described as a gripping or a sense of compression beneath the sternum. The pain occurring in cases of myocardial disease is of a different character from that of coronary origin, being mostly to the left of the mid-line and radiating to the region of the apex beat, or being felt only at the apex. It does not have the radiation to the back, neck or arm except when very severe, and is a more continued pain than the gripping paroxysmal pain of coronary disease. Its character is more aching even though it should be, as it sometimes is, very severe indeed. When these conditions cause pain there is nearly always objective dyspnoea, a very rare occurrence with coronary artery attacks.

The pain felt by neurotic patients and patients with hyperthyroidism is not in either case a mid-line pain, but lies to the left of the sternum, like the pain of cardiac disease, and is most often situated about the region of the apex beat. That these patients have typical areas of hyperalgesia may lead you to a stronger belief that organic disease is present, but you must not be deceived by this into a diagnosis of cardiac disease. These pains, like those of coronary disease, may be brought on or increased by exertion, but there is an irregularity of occurrence after exercise which is characteristic, and this taken with the lateral situation of the pain and the fact that it moves around, together with evidence of a neurotic make-up of the patient, should enable us to readily recognize the origin of the symptoms. It is important to remember that these pains do not occur with the milder forms of hyperthyroidism, so that the rapid pulse or tremor or some of the other signs or symptoms of Graves' disease will be pres-

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ent to help you. In looking over my records of cases of myocardial insufficiency I found fourteen cases that I have seen in the last year and a half in which the symptoms did not coincide with any of those above, and the true nature of the malady had not been suspected either by their physicians or by the patients themselves until the disease had progressed well toward a fatal termination. Nine of these patients have died and the other five are still under observation.

I have not included in these fourteen cases any case in which the symptoms attracted either the physician or the patient to the heart as the cause of the trouble, and wish to emphasize that none of these cases were considered heart cases until the disease was well progressed.

CASE REPORTS

Case I. Male 63—Had been a railroad official, now retired. Had complained for a number of years of indigestion. He had been treated by several physicians with no appreciable relief. He was generally advised that his malady was due entirely to his stomach and bowels and was dieted and given treatment along that line. One morning I happened to be at his home and was called into his room and found him deathly pale, covered with cold perspiration and unconscious. (This was the first time I had seen him professionally as he had been under the care of another physician.) He had arisen as usual in the morning and was feeling very well. He went into the bathroom to shave and suddenly fell and I was immediately asked to step in and see him. Upon examination his pulse was not felt at wrist and, listening over his heart, the heart beat was rapid and grossly irregular. As his pulse returned, a polygraphic tracing taken then showed the attack to be one of paroxysmal auricular fibrillation which soon subsided and the heart beat became perfectly regular except for an occasional premature auricular beat which soon disappeared, and I never found any irregularity in his pulse afterwards. He had no pain at any time and his only complaint was his indigestion and intestinal flatulence. He died in a neighboring city recently, and I will quote the following from a letter I received from his wife: "Mr. J. was very well all summer and we put off from month to month going down to see you. We had been out to Van Nuys all day, and after dinner he said: 'I feel exceptionally well tonight.' But a few minutes later gas began to bother him. I gave him soda but it didn't give him any relief. He had no pain in his heart but it beat very fast. He just said, 'I am so nervous, I can't stand it,' and became unconscious." I had this man under observation for some time after the first attack and outside of his heart being slightly enlarged there was nothing that would attract one's attention to it. The rate was normal, rhythm was regular. There were no abnormal sounds. There was no swelling of the feet, nor cough.

Case II. A man 54 years of age walked into my office complaining of pain in his stomach. He gave the following history: He had had a similar attack of indigestion, as he termed it, accompanied with severe pain while on the street two years ago and he was taken to the office of a chiropractor, who treated him for three weeks, during which time he was up and around. Since then he had been troubled a great deal with a sour stomach. He said that he ate too much, always having a good appetite. Under closer questioning he admitted having felt pain in the epigastrium after running, lifting, or going up hill. But all the time during the past two years he had done his work on his ranch and had been very well with the exception of his indigestion, which was always relieved immediately when he could get up the gas. Never had had any swelling of his feet; no cough except when he had a cold; never had rheumatism, tonsillitis or "flu," but his teeth were bad. He had had scarlet fever twenty years before, measles eight years before. On the day he consulted me he had carried two grips a considerable distance in Los Angeles and had taken the bus for San Diego. Soon after leaving Los Angeles he began to feel discomfort in his stomach, which increased, and when he arrived at Oceanside the discomfort had increased to such a degree that he went into the drug store to get something to make him vomit. After vomiting he felt better. He then stated that he knew it was his stomach, because he was always relieved after he could expel the gas. Examination revealed a well-nourished, well-muscled man, who had led an active outdoor life. His face was somewhat ashen, he was perspiring, held his hand over his epigastrium and complained of sense of fulness which was killing him and said if he could get something to expel the gas he would be all right, as he had had similar attacks before. His pulse was 84 regular, except for an occasional P. V. B. His B. pressure was 150 s. and 80 D. His apex was in the fifth interspace at the nipple line. The area of cardiac dullness extended 10½ cm. to the left of the median line and 3 cm. to the right. The first sound at the apex was split and second at apex was a trifle loud. At the base the aortic second was louder than the pulmonic. There were no murmurs anywhere except a slight after-tone to the aortic second heard at the ensiform. There were no rales to be heard in the chest. Urine S. S. showed a S. P. of 1026, no albumen, no casts. He was soon enough relieved to be taken home, although the distress never entirely ceased, and the next day there was some adema at the bases of the lungs, and he died suddenly while talking to his wife that evening. Post-mortem examination showed a heart somewhat enlarged, a thickened fibrous aorta with plagues, narrowed and sclerosed coronaries with complete occlusion of the left coronary by a thrombus.

Case III. Woman 60 years of age, who gave a history of abdominal symptoms for a number of years, always accompanied by gas on her stomach. She had been operated on for some pelvic disturbance and for appendicitis. She was now in the hospital to be operated on for a growth in the descending colon. I was called to examine her heart. I found a large, fleshy woman with some edema of her arms, giving a history of her feet swelling when she was up and around. She said she had a great deal of gas on her stomach and bowels, pain in her left side of abdomen, which had led up to where she was about to be operated for a growth in the descending colon. Upon examination, her pulse was 100, perfectly regular. B. P. 100, S. 70 D., area of cardiac dullness extended 11 cm. to the left and 4 cm. to the right of the median line. First sound at the apex was fairly good, the second at the apex was very faint, at the base the pulmonic second was much louder than aortic second. A faint systolic murmur was heard over the aortic second, but was not heard in the carotids. There were a few rales in both bases and a polygraphic tracing showed nothing except a variation in the output of the left ventricle. She said she felt quite well at present, except she was having a great deal of gas on her stomach. This patient was kept in bed and died suddenly about two weeks later, after excitement caused by transacting some important business. Dr. Thomson's report of post-mortem was as follows: Heart, large and moderately dilated, dark in color, right V. wall very thick, papillary muscles hypertrophied. Heart muscle very soft and friable, showed edema, was so soft that by a small amount of pressure the finger would go clear through. Chest contained fluid, both lower lobes of lung compressed. The descending colon was somewhat contracted, but showed no evidence of growth.

I have not the time to relate the histories of all these patients, but will give you a résumé of them. In eight cases the abdominal symptoms were accompanied at times with severe pain. Of this group of eight cases five of them had a blood pressure much below normal and in three the B. P. was slightly elevated. Six of this group are dead, all died very suddenly. Six of this group were males between the ages of fifty-four and seventy-six, and two were females between the same ages.

In none of these cases was there any valvular lesions of importance. The second group in which there were no attacks of pain, consisted of six patients, all males. Three of them had a low B. P. and in three of them the B. P. was slightly elevated. Three of this group are dead, all died rather suddenly according to reports; the exact termination I haven't been able to find out in all cases, as they died after leaving San Diego. Two of those that died had a low B. P. and in one it was elevated. Two of this group had pulsus alternans; in one the alternation followed a premature auricular beat, and in the other the alternation was continuous. The patient with continuous alternation is still alive, while the one with the short periods of alternation following the premature auricular beats is dead.

Of the whole series of fourteen cases only four of them showed a constant regular sequence of events in the cardiac cycle. Five of them showed periods of alternation in the polygraph tracing. Of these five three are dead and two are still living; of the remaining five cases one had an attack of paroxysmal auricular fibrillation; one had attacks of paroxysmal tachycardia with a short run of auricular flutter and is still alive, although he fell over in the park not long ago. Three showed only a premature V. beat, two of which are dead. Of the nine deaths all except two had some disturbance of the cardiac rhythm or pulsus alternans. In one of the cases with pulsus alternans, an electrocardognam showed a splintering of the Q. R. S. complex in lead 2.

Every one of the series had a heart enlarged to a point at or outside the nipple line, and eight of them had a B. P. below normal, six of whom are dead, while five of them showed a slightly elevated B. P., three of whom are dead. They all have this in common that months before the malignant nature of the malady had become apparent, mild gastric and abdominal symptoms had been complained of and the true nature of which had eluded some very competent observers.

The fourteen cases were all between the ages of fifty-four and seventy-eight, the greatest number being between the ages of fifty-four and sixty-five. There was no evidence of syphilis in any of them. None of them gave any evidence of gout, lead alcohol, nor the misuse of tobacco. I have not been able to trace any of these cases to a definite infectious origin. None of these cases were observed in my hospital work, all of them being observed in my private practice, which is the field par excellence for the observation of these cases.

The abdominal symptoms were the first signs of the myocardial insufficiency and none of them attributed any of their symptoms to their heart. In the mind of the patient all the symptoms were due to their stomach, otherwise why did the expulsion of gas give them relief. Some of them complained of fullness in the abdomen, having to loosen their clothing after eating and the escape of quantities of gas per rectum. The cardio-vascular symptoms vary considerably after the case is well advanced. The pulse may be slow and regular, of good quality, and may be thus maintained throughout an attack accompanied by severe pain. One died and the pulse never went above 80 and suddenly stopped while I was counting it.

The presence of a myocardial insufficiency may first betray itself by some irregularity of the pulse or very low B. P. or the discomfort or pain following some exertion or eating. As the case advances it may develop alternation flutter, premature ventricular beats, premature auricular beats, paroxysmal tachycardia or parox, auricular fibrillation; the B. P. is more often abnormally low than high. In the two cases of which I was able to get a post-mortem, in one there was distinct evidence of aortic and coronary sclerosis, while in the other there was a soft friable edemitus heart muscle through which one could push his finger with slight pressure, with no mention of coronary disease. I think in this type the pain, if there is any, is due to the exhaustion of the heart muscle rather than to an interference with its blood supply, as probably occurs in the type with the attacks of paroxysms of pain. Also this latter group in the interval between the attacks are able to get around and feel comparatively well, while in the group without the pain there was a progressive failure of the heart muscle, and finally the symptoms of cardiac dilatation with edema, and so forth.

The first evidence that there is a cardiac factor as well as an abdominal one may be a sudden drop of the systolic B. P. to a point around 100. There is one school who think there is really an abdominal angina associated with sclerosis of the abdominal arteries, and another school who lean rather to the view that the abdominal symptoms are reflexes from thoracic pathological processes.

The pathological explanation of even true angina is still in dispute. The true coronarians see a degenerative process of the coronary arteries in all cases, while Osler, Graham-Steele and others cite autopsies in which they could demonstrate no lesions of the coronary arteries. Allbutt finds the supragsigmoid area of the aorta the seat of the disease. As to the cause of the pain, the intermittent claudication theory of Allen Burns seems to satisfy many competent observers. It may be defined as a state in which the artery admits enough blood for quiet work but not enough for increased work, and a heart the coronary arteries of which are sclerosed and non-elastic can well bring about this state. On the other hand, Mackenzie holds that the symptoms in all cases are due to exhaustion of the heart muscle; and if I may add my humble opinion, I believe that the type with the paroxysmal attacks of pain are due to some interference with the blood supply to the heart muscle,

while in the other type the symptoms are due to exhaustion of a *diseased* heart muscle.

TREATMENT

The treatment of the two types is different in some respects. Both types are benefited by prolonged rest, mental and physical relaxation, and reduced alimentation. The iodides seem to exert a beneficial influence given over a long period of time. The group of cases characterized by progressive myocardial exhaustion without pain are benefited more by digitalis than are those of the other group. It has been my habit to restrict the proteins somewhat in the type with paroxysmal pain, as well as to diminish the starches in the form of potatoes, bread and cereals. At the same time definitely diminishing the amount of food intake, while in the other group of cases it is usually better to allow a liberal protein diet. I am guided in this by the percentage of urea retained in the blood. The fluid intake allowed depends upon the tendency to edema. The abdominal symptoms are very difficult to relieve at first, but they clear up readily as the heart muscle regains its efficiency. The paroxysmal pain of the one group is usually promptly relieved by the nitrites, while the discomfort and abdominal symptoms of the other group are usually made worse by its use. The greatest difficulty is experienced in convincing the patient what his trouble really is. It is difficult to convince a patient that he must restrict his activities because of indefinite abdominal or gastric symptoms and that his life is in danger because he belches a little or suffers from abdominal gas.

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RAILROAD SURGEONS CONDEMN ANTI-HEALTH AND ANTI-EDUCATION INITIATIVES

The following resolution was unanimously adopted by the Pacific Association of Railway Surgeons at their annual convention at San Francisco, August 26, 1922:

Whereas, There are three anti-health and anti-education initiatives before the people to be voted on at the general election, November 7, 1922, namely, the Chiropractic, Osteopathic and Anti-vivisection Initiatives; and,

Whereas, The three combined would permit the unqualified to practice unscientifically and prevent the qualified from practicing scientifically, by lowering educational standards, nullifying vital public health safeguards, making physicians and surgeons out of drugless practitioners with no proper preparation or experience; and,

Whereas, Educated physicians and surgeons would be "debarred forever" under the terms of one of these Initiatives, for daring to use a few guinea pigs to save the lives of countless people, be it.

Resolved, That this twentieth annual meeting of the Pacific Association of Railway Surgeons, in the interests of public health and safety and all whom we serve, do hereby condemn singly and collectively the Chiropractic, Osteopathic, Anti-vivisection Initiatives and urge all those interested in the scientific study, prevention, cure and control of diseases and the promotion of public health to vote against these dangerous measures.

THE GREAT SECOND TYPE OF CHRONIC ARTHRITIS.

By LEONARD W. ELY, M. D., San Francisco.

THIRD STUDY
(Synopsis of Previous Work.)

In several previous articles I have attempted to demonstrate that all cases of chronic arthritis can be divided into two great types or groups differing markedly in their clinical appearance, and radically in their X-ray picture and their pathological characteristics. The first type includes the bacterial infections, and has for its pathological feature a proliferative inflammation in the bone marrow, in the synovial membrane, or in both.

The second type differs sharply from the first in almost every way, and has received many names based on features which their originators deemed important. Among these names may be mentioned arthritis deformans, osteoarthritis, hypertrophic arthritis, degenerative arthritis, metabolic arthritis, senile arthritis, destructive arthritis. The disease has been variously ascribed to trauma, loosely to infection and to some mysterious error in metabolism, some unknown chemical substance floating in the blood stream. I have shown that the disease could not be caused by trauma. The morbid changes in the joint are as different from those caused by bacteria as black is from white, and our efforts at Stanford to obtain bacterial cultures from the bone marrow and from the joint fluid have been uniformly unsuccessful. In such a situation the easiest way out is to take refuge in words, and to say that the disease must be due to metabolic error, to a dyscrasia, to a diathesis, to autointoxication from the colon, to a displaced vertebra or to an error of mortal mind.

This disease is characterized clinically by the presence of bony and cartilaginous ridges at or about the lines of attachment of the capsule, revealed by the Roentgen film. The surgeon who operates upon an affected joint makes his diagnosis upon this production of bone and cartilage, and the pathologist in the laboratory, recognizing the bone and cartilage changes, also bases his diagnosis upon them, and has always maintained that they were the characteristic and fundamental feature of the disease.

I have shown that the changes in the bone and in the cartilage were not primary but were the result of a previous aseptic necrosis in the marrow in the vicinity of the joint. This necrosis affects the bone and the marrow, giving rise to larger or smaller sequestra, and later to cysts, to bone cavities and to the so-called ostitis fibrosa. I have tried to establish the connection of this disease with fracture of the neck of the femur in the aged, and with the arthritis which follows an intra-articular fracture in an elderly person. The whole appearance of the lesion of the bone pointed strongly to a non-bacterial infection as the cause, but up to a few months ago I had never been able to discover any clue as to the nature of this organism.

In two previous papers I had called attention to the coincidence of infection in the alveolar proc-